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Index terms:
Blood, flow dynamics, 957.12944
Intestines, ischemia, 70.761
Magnetic resonance (MR), oxygen transport, 957.12941, 957.761
Mesentery, ischemia, 792.266, 955.761
Magnetic resonance (MR), phase-contrast imaging, 957.12919
Magnetic resonance (MR), pulse sequences, 957.12919
Veins, mesenteric, 957.12944

Radiology 1998; 206:219–225

Abbreviations:
$\%Hbo_2$ = percentage of oxygenated hemoglobin
NOMI = nonocclusive mesenteric ischemia
SMV = superior mesenteric vein

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Author contributions:
Guarantor of integrity of entire study, K.C.P.L.; study concepts, K.C.P.L.; study design, L.R.P.; definition of intellectual content, K.C.P.L., L.R.P.; experimental studies, K.C.P.L., L.R.P., G.A.W.; data acquisition and analysis, K.C.P.L., L.R.P., S.P.; statistical analysis, K.C.P.L.; manuscript preparation and editing, K.C.P.L.; manuscript review, L.R.P., S.P., G.A.W.

Mesenteric Ischemia due to Hemorrhagic Shock: MR Imaging Diagnosis and Monitoring in a Canine Model[1]

PURPOSE: To test whether changes in the percentage of oxygenated hemoglobin ($\%Hbo_2$) and blood flow in the superior mesenteric vein (SMV), as measured with magnetic resonance (MR) imaging in vivo, can be used to diagnose and monitor mesenteric ischemia due to hemorrhagic shock in a canine model.

MATERIALS AND METHODS: Eight mongrel dogs (weight range, 20–30 kg) underwent fasting for 24 hours before the experiments. MR imaging measurements of SMV $\%Hbo_2$ and volume flow rate were obtained at the resting state and after 5%, 10%, and 15% of the blood volume of the dogs had been removed sequentially, which led to a total blood volume depletion of 30%. In four dogs, resuscitation was performed with normal saline solution in a volume equal to the total volume of blood removed.

RESULTS: SMV $\%Hbo_2$ and SMV flow measurements at the different stages of blood removal were all significantly different ($P < .05$) from baseline measurements and from each other. After volume replacement with normal saline solution, SMV $\%Hbo_2$ and flow were not significantly different ($P > .05$) from the baseline values.

CONCLUSION: SMV $\%Hbo_2$ and volume flow rate, as measured with MR imaging in vivo, can be used to diagnose and monitor mesenteric ischemia due to hemorrhagic shock in a canine model.

Nonocclusive mesenteric ischemia (NOMI), also called "low flow syndrome," "functional mesenteric infarction," or "spastic mesenteric insufficiency," is a condition associated with high morbidity and mortality (1–3). In NOMI, severe microvascular vasoconstriction results in intestinal ischemia even though macroscopic arterial blood flow is still present (1–3). NOMI is frequently associated with conditions that may compromise circulation, such as shock, hypotension, kidney diseases, atherosclerosis, diabetes mellitus, stroke, pancreatitis, hypertension, recurrent embolism, and polycythemia vera (1–3). NOMI is also frequently linked to the intake of drugs that may lower the mesenteric blood flow rate, such as digitalis, furosemide, ergotamine derivatives, and hydrochlorothiazide (1–3). In a large percentage of cases, the clinical presentation is ambiguous and often dominated by one of the underlying diseases. Mesenteric angiography is considered by most clinicians the only reliable diagnostic tool available today (1–3). Mesenteric angiography is invasive, however, and false-positive findings can occur in patients who are in shock, are receiving vasopressors, or have acute pancreatitis (1–3).

It has been demonstrated that in the superior mesenteric vein (SMV) the percentage of oxygenated hemoglobin ($\%Hbo_2$) and blood flow can be accurately measured in vivo by using flow-independent T2 measurements of the venous blood (4) and cine phase-contrast techniques (5), respectively. These techniques have been successfully used for studying chronic and acute occlusive mesenteric ischemia (6–13). The purpose of this study was to test the hypothesis that changes in $\%Hbo_2$ and blood flow in the SMV, as measured with magnetic resonance (MR) imaging in vivo, can be used to diagnose and monitor mesenteric ischemia due to hemorrhagic shock in a canine model.

TABLE 1
Summary of Data Obtained at Different Stages of Hemorrhagic Shock and Resuscitation

Parameter	Baseline	After Blood Loss			After Resuscitation with Saline Solution
		5%	15%	30%	
SMV %Hbo_2	73.7 ± 1.3 (8)	66.7 ± 2.1 (8)	59.3 ± 2.3 (8)	46.7 ± 3.6 (7)	70.1 ± 0.4 (4)
SMV flow (% of baseline)	100 (8)	76.3 ± 6.2 (8)	60.5 ± 6.1 (8)	37.9 ± 8.1 (6)	105.6 ± 9.6 (4)
Hematocrit (%)	39.5 ± 2.3 (8)	38.7 ± 1.8 (8)	37.3 ± 1.5 (7)	39.5 ± 1.7 (6)	30.1 ± 1.6 (4)
Systolic blood pressure (mm Hg)	86.1 ± 5.5 (7)	79.4 ± 3.8 (7)	67.0 ± 4.1 (7)	52.0 ± 4.9 (6)	100.3 ± 14.1 (4)

Note.—Data are reported as the mean ± standard error. Numbers in parentheses indicate the sample size in each category.

MATERIALS AND METHODS

In Vivo MR Oximetry

The details of the technique we employed for in vivo MR oximetry have been fully described previously (6,14). Before in vivo MR oximetry and cine phase-contrast MR imaging, axial and sagittal localizing images must be obtained to ensure that the imaging plane is perpendicular to the SMV. For accurate estimation of the T2 of blood in vivo, we modified the basic Carr-Purcell-Meiboom-Gill sequence to address the challenges of the in vivo environment. The resultant sequence has been described previously (6). For estimation of SMV %Hbo_2 in the animals, the sequence was used with the body coil and the following parameters: a repetition time of 2,000 msec and a time between 180° pulses of 12 msec with four echoes acquired at 30, 78, 126, and 222 msec (2,000/30, 78, 126, 222) on consecutive interleaves of the sequence; 20 spiral acquisitions per image; and two signals acquired. This yielded a total imaging time of 5 minutes 20 seconds. Section thickness was 7 mm, and in-plane resolution was 1.8 mm. Only a single section can be obtained with this technique. For the estimations of the T2 relaxation times, a region of interest was selected to be centered in the vessel with a diameter roughly equal to 50% of the maximum visible diameter. Typically, each region of interest covered 10–15 voxels measuring 2 × 2 × 7 mm each. To establish a quantitative relationship between the T2 of blood and %Hbo_2, a previously described in vitro calibration procedure was used (6,14). Blood samples were aerated to varying levels of %Hbo_2, as measured with a clinical reflectance oximeter (Oxicom 2100; Waters Instruments, Rochester, Minn) before and after the in vitro MR imaging studies. The T2 of the different blood samples was then measured by using the sequence described above. A least-squares fit of the data set of the T2 of blood and %Hbo_2 pairs was then used to convert the in vivo blood T2 measurements to the corresponding %Hbo_2.

TABLE 2
Matrix Summarizing the Correlations between the Different Physiologic Parameters

Parameter	Percentage of Blood Loss	SMV %Hbo_2	SMV Flow	Systolic Blood Pressure	Heart Rate
Percentage of blood loss	1.000	−0.862*	−0.748*	−0.728*	0.012
SMV %Hbo_2	−0.862*	1.000	0.661*	0.620*	−0.352
SMV flow	−0.748*	0.661*	1.000	0.510*	0.021
Systolic blood pressure	−0.728*	0.620*	0.510*	1.000	0.158
Heart rate	0.012	−0.352	0.021	0.158	1.000

Note.—SMV %Hbo_2 has the highest correlation to blood loss.
* Correlation is statistically significant ($P < .05$).

Cine Phase-Contrast Protocol

For the SMV flow-volume measurements, cine phase-contrast MR images were obtained at the level of the pancreatic head by using the following parameters: axial orientation, cardiac or peripheral gating, respiratory compensation, 24-cm field of view, 16 phases per cardiac cycle, 256 × 128 matrix, 25/10, 30° flip angle, two signals acquired, through-plane flow-encoding strength of 50 cm/sec, and section thickness of 5 mm.

Animal Preparation and Imaging Protocols

All animal procedures used in this study were approved by the Stanford University Institutional Animal Care and Use Committee. Eight mongrel dogs (weight range, 20–30 kg) were studied. The animals underwent fasting for 12 hours before the experiment and were premedicated with the subcutaneous administration of acepromazine maleate (Butler, Columbus, Ohio; dose, 0.01 mg per kilogram of body weight). Anesthesia was induced with the intravenous administration of thiopental sodium (Pentothal; Abbott Laboratories, North Chicago, Ill; dose, 25 mg per kilogram of body weight). The animals then underwent intubation and ventilation with an MR imaging–compatible ventilator (Omni Tech Medical, Topeka, Kan). Anesthesia was then maintained with 1.5%–2% halothane (Halocarbon Laboratories, River Edge, NJ). A catheter was inserted into the inferior vena cava via a femoral vein, and a model TC510 Mikro-tip catheter pressure transducer (Millar, Houston, Tex) was inserted into the aorta via the contralateral femoral artery. After catheter placement, the dogs were allowed to recover until all hemodynamic parameters reached a steady state. Then MR imaging was performed, which took an average of 15–20 minutes. All MR images were obtained with a 1.5-T whole-body imager (Signa; GE Medical Systems, Milwaukee, Wis) and a body coil. Axial and sagittal localizing images were obtained to ensure that the imaging plane was perpendicular to the SMV. MR imaging measurements of the T2 of blood and the volume flow rate in the SMV were then performed with the techniques described above.

After the baseline MR imaging measurements were performed, 5%, 10%, and 15% of the blood volume of the dogs was removed sequentially via the venous catheter, which led to a total blood volume depletion of 30%. Blood volume of the

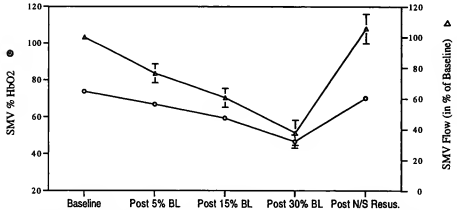

Figure 1. Plot of SMV $\%Hbo_2$ and SMV blood flow (expressed as percentage of baseline flow for standardization) at baseline, at the different stages of blood loss (*BL*), and after resuscitation with normal saline solution (*Post N/S Resus*). Some error bars are too small to be shown on this scale.

dogs was estimated at 86.2 mL/kg (15). In four dogs, resuscitation was performed by injecting normal saline solution via the venous catheter into the inferior vena cava in a volume equal to the total volume of blood removed. MR imaging measurements of the T2 of blood and the volume flow rate in the SMV were repeated after each occasion of blood removal and after resuscitation. MR imaging measurements were obtained approximately 15 minutes after each blood removal and after resuscitation. The MR imaging measurements took approximately 15 minutes to acquire. During each session of blood removal and after resuscitation, 20 mL of the blood sample was citrated and then aerated to varying levels of $\%Hbo_2$. These blood samples were then used for the in vitro calibration of the T2 of blood versus $\%Hbo_2$ as described above.

Statistical Analysis

MR imaging measurements of SMV $\%Hbo_2$ and blood flow at the different stages of blood removal and after resuscitation were compared with the baseline measurements and with each other by using the Student paired *t* test. A correlation matrix of the physiologic parameters (blood loss, SMV $\%Hbo_2$, SMV flow, systolic blood pressure, and heart rate) was also computed by using a standard statistics software package (STATVIEW 4.01; Abacus Concepts, Berkeley, Calif).

RESULTS

The quantitative data obtained are summarized in Table 1. MR imaging measurements of SMV $\%Hbo_2$ at the different stages of blood removal were all significantly different ($P < .05$) from the baseline measurements and from each other. These findings are graphically illustrated in Figure 1. After resuscitation with normal saline solution, SMV $\%Hbo_2$ was not significantly different ($P > .05$) from the baseline measurement. Similarly, SMV flow values at the different stages of blood loss, expressed as percentages of the baseline flow for standardization, were all significantly different ($P > .05$) from the baseline measurement and from each other (Fig 1). After resuscitation, SMV flow became slightly higher than but not significantly different ($P > .05$) from the baseline measurement. Baseline SMV blood flow varied from 96.8 to 209.4 mL/min (mean ± standard error, 158.9 mL/min ± 14.9). MR images obtained from a typical experiment are illustrated in Figure 2. A typical in vitro calibration curve of the T2 of blood versus $\%Hbo_2$ is illustrated in Figure 3.

Hematocrit at the different stages of blood removal was not significantly different ($P > .05$) from the baseline. After resuscitation with normal saline solution, however, hematocrit was significantly lower ($P < .05$) than baseline. This finding is graphically illustrated in Figure 4. Systolic blood pressure, on the other hand, decreased significantly ($P < .05$) after 15% blood loss (Fig 4). After resuscitation, systolic blood pressure was restored to the baseline level. The relatively low baseline systolic blood pressure was attributed to the anesthesia.

The correlation matrix between the different physiologic parameters is shown in Table 2. Blood loss (as a percentage) had the highest correlation with SMV $\%Hbo_2$, followed by SMV flow and systolic blood pressure. All these correlations were statistically significant ($P < .05$). Heart rate, on the other hand, had no significant correlation with any other physiologic parameter. The intestine-specific physiologic parameters (SMV $\%Hbo_2$ and SMV flow) were more highly correlated with each other and with blood loss and were less correlated with systolic blood pressure, even though all these correlations were statistically significant.

One dog died after 30% of blood was removed and before MR imaging measurements could be obtained. Another dog died after 30% of blood was removed and MR SMV $\%Hbo_2$ was measured but before SMV flow was measured. Resuscitation was not administered in two other dogs owing to technical difficulties and imaging time limitations. Some blood pressure and hematocrit data are missing because the catheter pressure transducer was not working during one experiment and one blood sample was accidentally discarded before hematocrit was measured after 15% of the blood volume had been removed from one dog.

DISCUSSION

NOMI is associated with up to 50% of all mesenteric infarctions found at autopsy (16) and accounts for approximately 17% of all cases of acute mesenteric ischemia (1). In recent years, the incidence of NOMI is thought to have declined owing to the more extensive use of systemic vasodilators such as calcium channel blockers and nitrates (1). NOMI is still considered, however, an underdiagnosed disease in intensive care medicine (1). NOMI is most often found in critically ill elderly patients who have markedly diminished cardiac output, have hypovolemia, or are receiving vasoconstrictive medications (16). The median age of patients in whom NOMI is diagnosed is 75 years (16).

The exact pathophysiology of NOMI is still not well understood (1). A systemic "low-flow" state and mesenteric vasoconstriction, however, are important factors that are common to all NOMI cases (1,17). It is believed that normal mesenteric vessels constrict during a systemic low-flow state, so that more blood can be provided for the more essential organs and tissues, such as the brain, myocardium, and kidneys (17). If mesenteric vasoconstriction is prolonged, NOMI may persist even after the primary cause is corrected (1,18). Lowering the perfusion pressure in the mesenteric circulation relaxes the "pre-

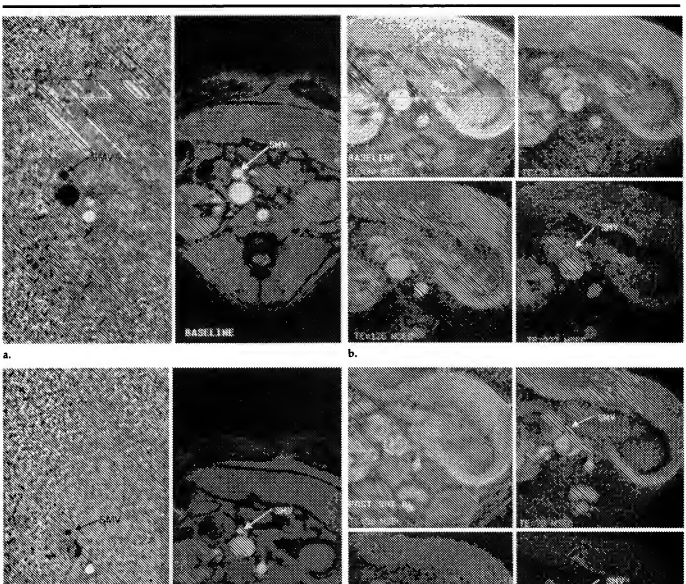

Figure 2. MR images from a typical experiment. **(a)** Peak systolic cine phase-contrast velocity (left) and magnitude (right) images (25/10, 30° flip angle) obtained at baseline. **(b)** Images (2,000/30, 78, 126, 222) obtained from the in vivo oximetry sequence at baseline. Even at an echo time (*TE*) of 222 msec, the signal intensity of SMV blood is still fairly high, which indicates a long T2 relaxation time and high $\%Hbo_2$. **(c)** Peak systolic cine phase-contrast velocity (left) and magnitude (right) images (25/10, 30° flip angle) obtained after 30% blood loss (*BL*). Notice a marked decrease in the size of the SMV on the magnitude image and a decrease in the darkness of the SMV blood on the velocity image, which indicate a marked decrease in SMV blood flow as compared with baseline. **(d)** Images (2,000/30, 78, 126, 222) obtained from the in vivo oximetry sequence after 30% blood loss. Notice a decrease in signal intensity in the SMV blood on the late echo images, which indicates a decrease in SMV $\%Hbo_2$. (*Fig 2 continues.*)

capillary" sphincters so that more intestinal villi are perfused (19). This relaxation of precapillary sphincters leads to an increase in the area for countercurrent exchange. In addition, the transit time for plasma in the whole villus also increases from 5 to 15–20 seconds (19). The most important result of these alterations in countercurrent exchange is that more oxygen is extracted in the proximal end of the villi, which leads to decreased $\%Hbo_2$ at the tips of the villi. Eventually,

e. f.

Figure 2 *(continued).* **(e)** Peak systolic cine phase-contrast velocity (left) and magnitude (right) images (25/10, 30° flip angle) obtained after normal saline resuscitation (*POST NS RESUS*). Notice that the size of the SMV and the velocity of SMV flow are much higher than those after 30% blood loss and similar to those of baseline. **(f)** Images (2,000/30, 78, 126, 222) obtained from the in vivo oximetry sequence after normal saline resuscitation. Notice that the signal intensity of SMV blood on the late-echo images is higher than that after 30% blood loss and is closer to that at baseline.

mucosal ulcerations develop at the villus tips (16–19). Intestinal mucosal damage leads to the release of cardiotoxic material, which can further aggravate systemic hypotension and NOMI (19).

To decrease the morbidity and mortality of NOMI, early diagnosis is of paramount importance. If damage is limited to mucosa and submucosa at the time of diagnosis and intervention, full recovery is possible. If muscularis and serosa are involved, intestinal infarction with associated high mortality will result (1). A number of laboratory tests have been used to diagnose NOMI, but none of them has been found to be sensitive or specific enough. Currently, mesenteric angiography remains the only reliable tool for the early diagnosis of NOMI. The sensitivity and specificity of angiography, however, have also been questioned. First, mesenteric vasoconstriction in patients who are in shock, have acute pancreatitis, or are receiving vasopressors does not necessarily imply NOMI (1). Second, angiographic contrast material can act as a vasodilator, so vasoconstriction can be underdiagnosed at angiography (17). Third, the major resistance vessels in the mesenteric circulation are the precapillary arterioles, which cannot be seen on angiograms (17). Since intestinal hypoperfusion and increase in oxygen extraction are the pathophysiologic hallmarks of NOMI, noninvasive monitoring of these parameters should theoretically allow us to diagnose and monitor the course of NOMI effectively.

In our series, after 5% blood loss, the SMV flow decreased by 24% and mean SMV $\%Hbo_2$ decreased by 7%, which indicated that these MR imaging measurements are sensitive to even a mild degree of hypovolemia. Removal of 30% of the blood volume resulted in the deaths of two of the eight animals in our series, which indicated that we approached the point of maximal stress in our model. At 30% blood loss, SMV blood flow decreased by 62% and mean SMV $\%Hbo_2$ decreased by 27%. After resuscitation with normal saline solution, SMV blood flow was higher than the baseline value but SMV $\%Hbo_2$ was lower than the baseline values. It has been well documented that in hemorrhagic shock, mesenteric blood flow usually increases above the baseline level after retransfusion (19). This finding is attributed to an accumulation of "local metabolites," but it may also be due to a depletion of neurotransmitter in the vasoconstrictor fibers (19). Since the oxygen-carrying capacity of blood is lower after resuscitation with normal saline solution, as indicated by the lower hematocrit, it is interesting to observe that the SMV $\%Hbo_2$ is only slightly lower than baseline after resuscitation. The reason that the postresuscitation SMV $\%Hbo_2$ is not much lower than the baseline value is because oxygen uptake is the product of the difference between arterial and venous Hbo_2 and blood flow. Since the postresuscitation SMV blood flow is higher than the baseline blood flow, the intestinal oxygen uptake can approach the baseline value with SMV $\%Hbo_2$ only slightly lower than baseline, even though the oxygen-carrying capacity of the blood is lower after normal saline resuscitation. It will be interesting to use this model for studying the response of SMV $\%Hbo_2$ to different resuscitation fluids, which include various artificial blood products that potentially can expand the intravascular volume for a prolonged period without markedly lowering the oxygen-carrying capacity of blood.

Several limitations in our study may influence how these techniques can be applied clinically. First, even though the canine model has been most extensively used for studying mesenteric ischemia,

there are interspecies differences in mesenteric vascular physiology among dogs, cats, monkeys, and human beings (19). Second, anesthetic agents used during the experiments may alter the hemodynamic responses to hemorrhagic shock both systemically and in the mesenteric circulation. Third, a baseline measurement of SMV blood flow and $\%Hbo_2$ is not available in most clinical situations, so comparisons with average normal values are required for establishing the diagnosis of NOMI. This limitation may decrease the sensitivity and specificity of these techniques, since normal fasting SMV blood flow varies widely among patients (11). This finding is again confirmed by the wide variation of baseline SMV blood flow recorded in this experiment. In our experience, the variation in normal fasting SMV $\%Hbo_2$ is less than that in SMV blood flow (7); therefore, in vivo MR oximetry may be more sensitive and specific than SMV blood flow measurements in diagnosing NOMI. Both techniques, however, should be useful for monitoring the course of NOMI after treatment that may have consisted of correction of underlying disease and intra-arterial infusion of vasodilators into the mesenteric circulation. This is indicated because the correlations between blood loss and the intestine-specific physiologic parameters (SMV $\%Hbo_2$ and SMV flow) are higher than correlations between blood loss and the systemic physiologic parameters (systolic blood pressure and heart rate) in our experiment. In addition, since the correlations between the systemic physiologic parameters and the intestine-specific parameters are not high, it will be more difficult to diagnose NOMI due to hemorrhagic shock with systemic physiologic data alone. Fourth, the pulse sequence we use for in vivo oximetry is not widely available, but other techniques such as specially designed gradient-echo imaging have been successfully used for this purpose (20). Therefore, any groups with expertise in pulse sequence designs can potentially develop various techniques useful for in vivo oximetry.

In conclusion, our results demonstrate that SMV $\%Hbo_2$ and volume flow rate, as measured with MR imaging in vivo, can be used to diagnose and monitor mesenteric ischemia due to hemorrhagic shock in a canine model.

Practical application: These initial results suggest that MR imaging may be an accurate, sensitive, and noninvasive method for the diagnosis of acute NOMI due to hypovolemic shock or, possibly, other causes. Further investigations of NOMI in other animal models and in patients are warranted.

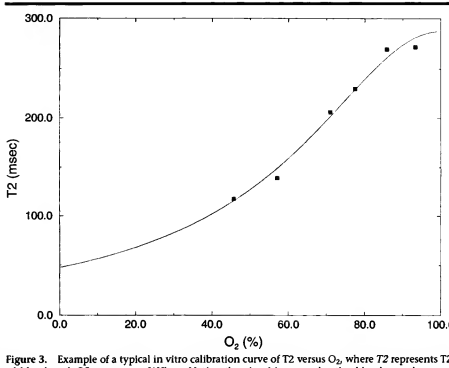

Figure 3. Example of a typical in vitro calibration curve of T2 versus O_2, where *T2* represents T2 of blood and *O2* represents $\%Hbo_2$. Notice that in this example, the blood samples were oxygenated to six different saturation levels and the curve was fitted to all six T2-O_2 data pairs. By using this calibration curve, the in vivo SMV blood T2 measurements can be converted to estimates of $\%Hbo_2$.

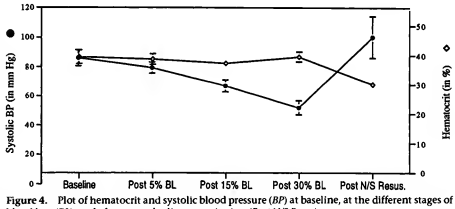

Figure 4. Plot of hematocrit and systolic blood pressure (*BP*) at baseline, at the different stages of blood loss (*BL*), and after normal saline resuscitation (*Post N/S Resus*).

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